

Effects of pacing site on QRS morphology in Wolff-Parkinson-White syndrome

With special reference to 'pseudo-tachycardia-dependent block in accessory pathway' and 'atrial gap'

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In a patient with a Wolff-Parkinson-White (WPW) syndrome type A mid-right atrial stimulation at a rate of 73/min produced a lesser degree of ventricular pre-excitation than when a slower sinus rhythm was present. This paradoxical effect was not related to tachycardia-dependent block in the accessory pathway because pre-excitation again increased at faster pacing rates. It was partly the result of a (proportionally) greater prolongation of intra-atrial conduction time to the accessory pathway than to the atrioventricular node and partly of a faster atrioventricular nodal conduction time. The latter, in turn, could be attributed either to later-than-normal arrival of excitation at the atrioventricular node, at a time when this structure was more recovered, or to a change in the site or mode of entry into the atrioventricular node.

A gap in the atria was present because at a St1-St2 interval shorter than that at which A2 had been blocked in the accessory pathway conduction was again possible, but with longer A1-A2 intervals. Finally, at similar, short, coupling intervals the impulse penetrated the atrioventricular node from the mid-right atrium but not from the coronary sinus. The unusual findings in this case support a recent assumption that in patients with WPW type A atrial stimulation should be performed from the coronary sinus to minimize the potential sources of error which can be produced by intra-atrial delay.

Although the effects of rate on QRS morphology in the WPW syndrome are well known (Gallagher *et al.*, 1975; Wellens, 1975), the results produced by changing the pacing site have not received much attention. Several authors recognized that conduction time from pacing site to accessory pathway (AP) played an important role (Gallagher *et al.*, 1975; Wellens, 1975; Touboul *et al.*, 1973; Wellens, Schuilenburg, and Durrer, 1971; Zipes, Rothbaum, and DeJoseph, 1974; Denes *et al.*, 1974). When the pacing site was close to the AP the pre-excited area was larger than when the site was far away from the AP. In this report we show that the site of stimulation can also influence QRS morphology in the WPW syndrome by exposing an intra-atrial conduc-

tion defect (Wellens *et al.*, 1971; Batsford *et al.*, 1974; Castellanos *et al.*, 1970) and probably by altering the moment of arrival of excitation at the atrioventricular node (Castellanos *et al.*, 1970) or by changing the site and mode of entry in this structure (Batsford *et al.*, 1974; Aranda *et al.*, 1976).

Case report

A 48-year-old man with the WPW syndrome and repetitive supraventricular tachyarrhythmias was referred to the cardiovascular laboratory for electrophysiological evaluation. His bundle recordings and electrical stimulation were performed after explaining the procedure and obtaining consent (Castellanos *et al.*, 1970; Aranda *et al.*, 1976).

The left-sided panel in Fig. 1 was obtained during

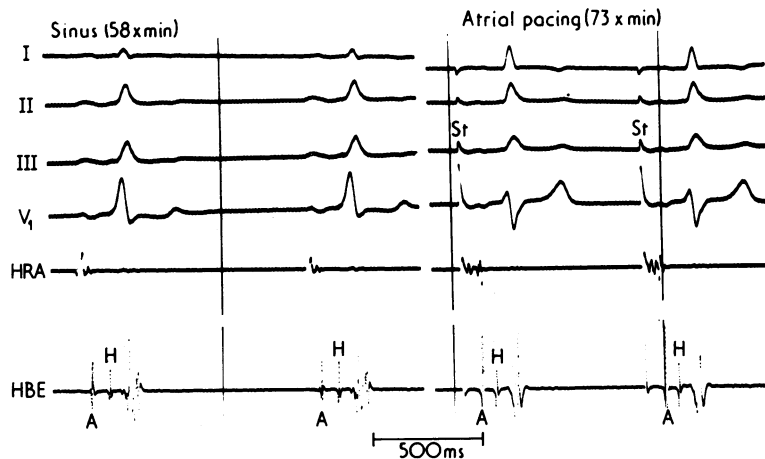


FIG. 1 Sinus rhythm (rate 58/min) with WPW type A morphology (left). During mid-right atrial stimulation at faster rate (73/min) degree of QRS distortion and bizarreness was less than when sinus rhythm was present. However, ventricular complexes probably still showed some degree of pre-excitation as determined by comparison with QRS morphology resulting from exclusive conduction through normal (atrioventricular node-His-Purkinje) pathway (see end of Fig. 6). Less pre-excitation at faster rates might, at first glance, be attributed to tachycardia-dependent block in the accessory pathway. HRA=high right atrium; HBE=His bundle electrograph; A=atrial electrogram recorded in vicinity of atrioventricular node; H=His bundle electrogram; St=pacemaker stimulus artefact.

sinus rhythm at a rate of 58/min (cycle length 1030 ms). The wide QRS complex showed an initial slurring with a predominantly positive deflection in V₁ (WPW type A). The PR and AH intervals measured 125 and 90 ms respectively. The H deflection was inscribed at the onset of ventricular depolarization. Thus the QRS complex was most probably a fusion beat resulting from ventricular activation through both normal (atrioventricular node-His-Purkinje) and accessory atrioventricular pathways. The PA and PH intervals lasted 35 and 125 ms respectively.

Fig. 1, right, was obtained during septal mid-right atrial (MRA) pacing at a rate of 73/min (cycle length 820 ms). The corresponding intervals were of the following duration: St-A, 90 ms; AH, 60 ms; St-H, 150 ms; HV, 40 ms, and St-V, 190 ms. The QRS complexes were narrower and showed a lesser degree of pre-excitation. Though the slightly slurred r wave was smaller than the S wave in lead V₁ the r/S ratio in this lead was still greater than in beats resulting from exclusive atrioventricular node-His-Purkinje conduction (end of Fig. 6). This faster rate of decrease in the degree of pre-excitation was unexpected, since it is known that in the WPW syndrome an increase in rate generally produces a greater amount of pre-excitation.

The unusual findings in Fig. 1, right, may be explained by assuming that the impulse traversing the atrioventricular node-His-Purkinje pathway was responsible for activating a greater part of the ventricles than during sinus rhythm. This, in turn, probably reflected the fact that intra-atrial conduction time appeared

to have been longer (from the paced site) to the AP than to the atrioventricular node. However, other factors such as a shortening of atrioventricular nodal conduction time (given by a decrease in the AH interval from 90 ms to 60 ms) might have also played a role (see Discussion).

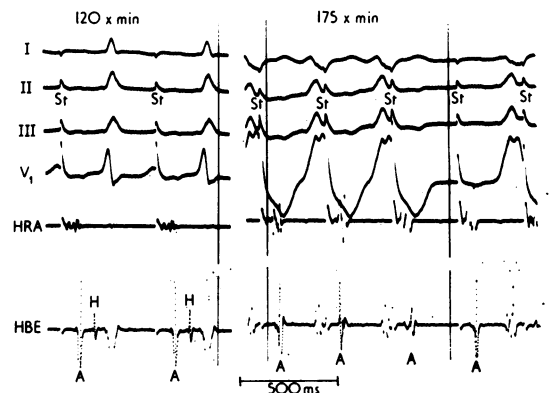


FIG. 2 During mid-right atrial stimulation at rate of 120/min (left) degree of pre-excitation increased (as compared to when rate was 73/min), indicating that tachycardia-dependent block in accessory pathway was not responsible for decrease in pre-excitation observed in Fig. 1. Right-sided panel shows QRS complexes resulting from exclusive accessory pathway conduction.

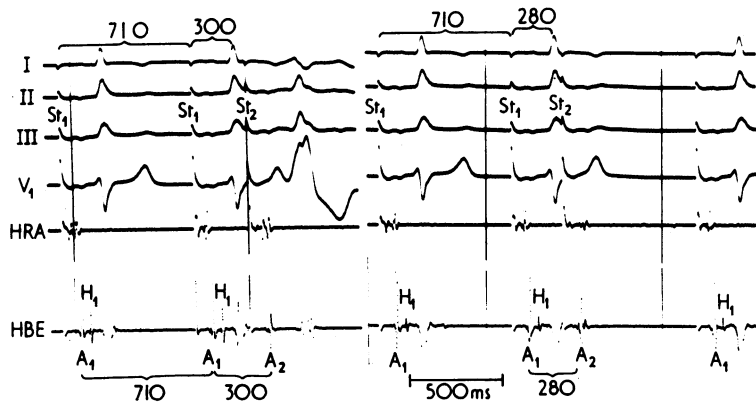


FIG. 3 Mid-right atrial stimulation at St1-St2 and A1-A2 intervals of 300 ms (left). A2 reached ventricles with QRS morphology attributed to exclusive accessory pathway conduction. At St1-St2 and A1-A2 intervals of 280 ms A2 was blocked in both normal and accessory pathways (right). St1 and St2=driving and testing stimuli, respectively; A1 and H1 and A2 and H2=right atrial and His bundle deflections produced by driving and testing stimuli, respectively. All values in ms.

At a stimulation rate of 120/min (Fig. 2, left) QRS morphology again resembled that seen during sinus rhythm. This occurred at a cycle length (500 ms), which was almost half that when sinus rhythm was present. The conduction intervals had the following values: St-A, 90 ms; AH, 90 ms; St-H, 180 ms; HV 10 ms, and St-V, 190 ms. Since the St-A and St-V intervals had the same values as those in Fig. 1, right, it was assumed that the intra-atrial conduction time did not increase. The greater degree of pre-excitation compared with when the rate was 73/min may be explained by the rate-related increase in atrioventricular nodal conduction time.

The right-sided panel in Fig. 2 shows the morphology

that appeared to have resulted from exclusive AP conduction. At a rate of 175/min the H deflection was lost within the ventricular electrogram of the His bundle electrographic lead. The third stimulus artefact (St) produced an atrial deflection which was blocked in both normal and accessory pathways.

Pacing with the extrastimulus method (Wit *et al.*, 1970) from a mid-right atrial site (MRA) was performed at a basic cycle of 710 ms. Driven QRS complexes thus showed a lesser degree of pre-excitation than when sinus rhythm was present. At coupling intervals between 750 and 320 ms the St1-St2, A1-A2, and V1-V2 intervals were similar. When St1-St2 and A1-A2 measured 310 ms and 300 ms (Fig. 3, left), H2 was lost within the

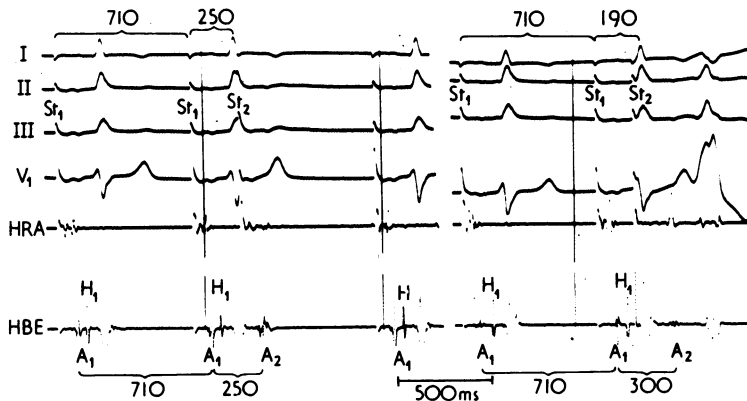


FIG. 4 At St1-St2 and A1-A2 intervals of 250 ms A2 was blocked in both normal and accessory pathways (left). At St1-St2 interval of 190 ms A1-A2 measured 300 ms. A2 was conducted because it was sufficiently delayed in atria so as to reach accessory pathway after end of its effective refractory period (atrial gap).

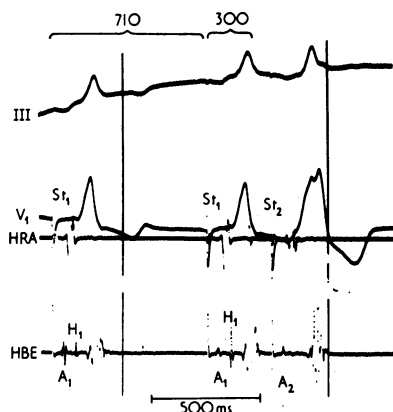


FIG. 5 Coronary sinus stimulation at same cycle length (710 ms) as that at which mid-right atrium was paced (Fig. 3 and 4) showing greater pre-excitation. Exclusive accessory pathway conduction occurs at St1-St2 and A1-A2 interval of 300 ms.

ventricular electrogram (V2) recorded by the HBE lead. The QRS morphology corresponded to that attributed to exclusive AP conduction. At St1-St2 and A1-A2 intervals between 280 and 250 ms A2 was not followed by either H2 or V2 (Fig. 3, right, and Fig. 4, left). This occurred because A2 reached the atrioventricular node and AP during their effective refractory period.

At St1-St2 intervals ranging between 230 and 190 ms the A1-A2 intervals again increased to 300 ms. Con-

duction to the ventricles was therefore possible almost exclusively through the AP with V1-V2 intervals of 320 ms (Fig. 4, right). Since this occurred with St1-St2 intervals shorter than those at which atrioventricular conduction had failed, it seems that there was an atrial gap (Wu *et al.*, 1974) between the paced site and the entrance to the AP. The difference between St1-St2 and A1-A2 intervals ($300-190=110$ ms) suggests that St2 was significantly more delayed in the atria than St1. Finally, the effective refractory period of the atria was reached at a St1-St2 interval of 180 ms.

Pacing with the extrastimulus technique at the same driving cycle length (710 ms) from the coronary sinus (CS) showed that QRS complexes produced by St1 had a greater degree of pre-excitation than when paced from the MRA (Fig. 5). The St-V (100 ms) and St-H (100 ms) intervals were shorter than during MRA driving (Fig. 3 and 4). At St1-St2 intervals from 750 to 290 ms both A1-A2 and V1-V2 intervals were the same (Fig. 5).

However, when the St1-St2 and A1-A2 intervals were reduced to 280 ms (Fig. 6) A2 was blocked in the AP and conducted exclusively through the atrioventricular node-His-Purkinje pathway with a normal (50 ms) HV interval and narrow QRS complex to initiate a run of reciprocating atrioventricular tachycardia. Note that at similar St1-St2 and A1-A2 intervals A2 had been unable to penetrate the atrioventricular node when the MRA was paced (Fig. 3, right). This explains why the tachycardia was elicited only when the CS was paced.

In this patient CS stimulation was not performed with St1-St2 intervals of less than 270 ms because St2 consistently produced long lasting runs of tachycardia.

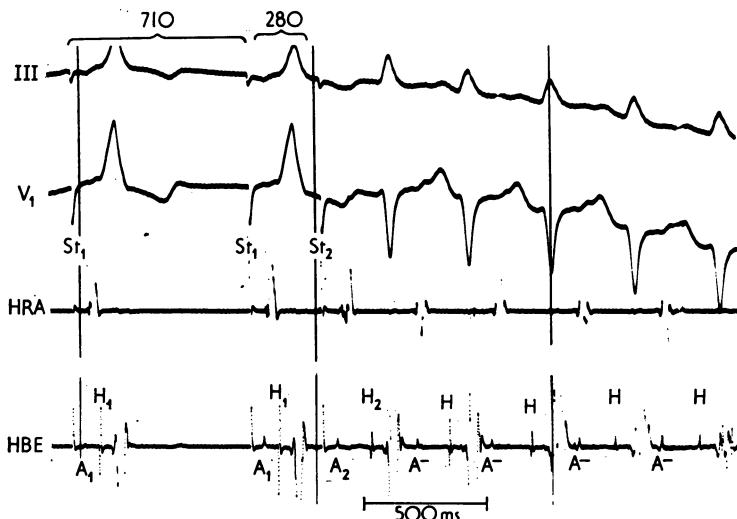


FIG. 6 Reciprocating atrioventricular tachycardia triggered by premature stimulus delivered to coronary sinus. Arrhythmia occurred at A1-A2 interval (280 ms) at which atrioventricular nodal conduction had not been possible during mid-right atrial pacing (Fig. 3, right). A⁻ = retrograde atrial deflections.

Discussion

Several factors are responsible for the different QRS morphologies seen in patients with the WPW syndrome. With the same site of stimulation an increase in atrial rate usually produces a greater degree of pre-excitation because the pacing-induced atrioventricular nodal delay decreases the amount of ventricular muscle depolarized by the impulse traversing the normal (atrioventricular node-His-Purkinje) pathway (Castellanos *et al.*, 1970; Wellens *et al.*, 1971; Wellens and Durrer, 1975).

At a constant rate changing the pacing site influences QRS morphology because of differences in conduction time between the area of impulse initiation and the atrial end of the AP. Thus in WPW type A CS stimulation results in shorter St-V intervals and wider QRS complexes (Fig. 1) than does right atrial pacing (Fig. 5). The reverse occurs in WPW type B—that is, the St-V intervals and QRS complexes are shorter and wider, respectively, from the right atrium than from the CS (Touboul *et al.*, 1973; Denes *et al.*, 1974). In addition, at a constant rate the pacing site may also influence QRS morphology by altering the moment of arrival of excitation at the atrioventricular node (Castellanos *et al.*, 1970) and by changing the site and/or mode of entry into the atrioventricular node (Batsford *et al.*, 1974; Aranda *et al.*, 1976). For example, in a patient with WPW type B reported by Castellanos *et al.* (1970) two different QRS patterns and alternating St-A intervals were seen at a pacing rate of 155/min. Narrow ventricular complexes and (presumably) shorter AH intervals occurred when the St-A intervals had a value of 100 ms compared with when they measured 50 ms. Moreover, Batsford *et al.* noted that in 50 per cent of their patients with normal PR intervals in whom high right atrial and CS pacing were performed atrioventricular nodal conduction and refractoriness appeared to have been faster and shorter, respectively, from the CS than from the high right atrium (Batsford *et al.*, 1974).

In a given case the variations in QRS morphology observed by changing the site and/or rate of pacing are the result of an interplay of factors. For example, the decrease in the amount of pre-excitation during MRA stimulation at a rate of 73/min (as compared with when sinus rhythm was present at a rate of 58/min) did not result from the existence of an accessory pathway with a long effective refractory period (tachycardia-dependent block in the accessory pathway), since the QRS complexes again became wider at even faster rates (Fig. 2).

The unusual response seen in Fig. 1, right, suggests that the impulse traversing the normal

pathway contributed more to ventricular depolarization than that propagating through the AP. This could have resulted in part from a proportionally greater increase in intra-atrial conduction time to the accessory pathway than to the atrioventricular node and in part from a faster atrioventricular nodal conduction time. The latter, in turn, might have reflected the pacing-induced, relatively late arrival of excitation at the atrioventricular node at a moment when this structure was more recovered (Castellanos *et al.*, 1970), or a change in the site or mode of entry into the atrioventricular node (Batsford *et al.*, 1974; Aranda *et al.*, 1976).

On the other hand, at faster pacing rates (Fig. 2) the rate-related atrioventricular nodal delay (given mainly by the prolongation of the AH interval) became a more important factor than the intra-atrial conduction time to the AP. Thus the impulse propagating through the atrioventricular node-His-Purkinje pathway contributed less to ventricular depolarization. An atrial gap (Wu *et al.*, 1974) was exposed during MRA pacing with the extrastimulus technique, since the premature stimulus delivered to the atria could activate the ventricles *exclusively* through the AP at shorter coupling intervals than those at which it had been unable to do so (Fig. 3 and 4). This was seen at a St1-St2 interval of 190 ms because the wavefront produced by St2 was sufficiently delayed in the atria by an increase in latency or conduction time, or both. Therefore it reached the AP after the end of its effective refractory period.

This type of gap occurred only between the atria and the AP because conduction was not resumed through the atrioventricular node-His-Purkinje pathway. According to the classical mechanism used to explain the gap phenomena the effective refractory period of the AP must have been longer than the functional refractory period of the atrium (Wu *et al.*, 1974). However, the relatively long time that the impulse had to traverse the atria early in the relative refractory period also favoured the occurrence of the gap.

In the absence of a better measurement it is customary to plot A1-A2 intervals against V1 (delta 1)-V2 (delta 2) intervals when determining the AP's effective refractory period (Castellanos *et al.*, 1973). Yet it should be understood that an atrial electrogram recorded from close to the entrance to the AP may, especially in WPW type A, give more valid results than an electrogram recorded from the right atrium in the vicinity of the atrioventricular node.

Pacing with the extrastimulus technique from the CS showed that at the same driven cycle length as that from the MRA the QRS complex showed

greater pre-excitation. Since the AH interval had similar values, this may be considered to be the result of the shorter conduction time between the CS and atrial end of the AP. The shorter time that the impulse had to reach the AP during the relative refractory period also explains why an atrial gap was not seen during CS stimulation.

Another interesting phenomenon was that at short coupling intervals the premature impulse delivered to the CS activated the His bundle through the atrioventricular node at an A1-A2 interval at which from the MRA it had been unable to do (Fig. 3, right, and Fig. 6). Because this was the interval at which reciprocating tachycardias occurred, the arrhythmias were therefore triggered only by CS stimuli. Again, as mentioned previously, differences in the place of impulse initiation may have produced a change in the site and/or mode of entry into the atrioventricular node.

Finally, the findings in this case support a recent statement that CS stimulation is superior to right atrial pacing in the evaluation of patients with WPW type A (Gallagher *et al.*, 1975; Wellens and Durrer, 1975). However, the effects of right atrial stimulation in these cases still require further study.

References

- Aranda, J., Castellanos, A., Jr., Moleiro, F., and Befeler, B. (1976). Effects of the pacing site on A-H intervals and refractoriness in patients with short P-R intervals. *Circulation*, **53**, 33.
- Batsford, W. P., Akhtar, M., Caracta, A. R., Josephson, M. E., Seides, S. F., and Damato, A. N. (1974). Effect of atrial stimulation site on the electrophysiological properties of the atrioventricular node in man. *Circulation*, **50**, 283.
- Castellanos, A., Chapunoff, E., Castillo, C., Maytin, O., and Lemberg, L. (1970). His bundle electrograms in two cases of Wolff-Parkinson-White (pre-excitation) syndrome. *Circulation*, **41**, 399.
- Castellanos, A., Jr., Myerburg, R. J., Craparo, K., Befeler, B., and Agha, A. S. (1973). Factors regulating ventricular rates during atrial flutter and fibrillation in pre-excitation (Wolff-Parkinson-White) syndrome. *British Heart Journal*, **35**, 811.
- Denes, P., Wyndham, C., Dhingra, R., Wu, D., Amat-y-Leon, F., Levitsky, S., and Rosen, K. (1974). Prediction of anomalous pathway location in patients with WPW syndrome using atrial pacing at multiple sites. *Circulation*, **50**, Suppl. 3, 222.
- Gallagher, J. J., Gilbert, M., Svenson, R. H., Sealy, W. C., Kasell, J., and Wallace, A. G. (1975). Wolff-Parkinson-White syndrome. The problem, evaluation and surgical correction. *Circulation*, **51**, 767.
- Touboul, P., Clément, C., Porte, C., Chuliat, J. C., Bons, J. P., and Delahaye, J. P. (1973). Étude comparée des effets de la stimulation auriculaire gauche et droite dans le syndrome de Wolff-Parkinson-White. *Archives des Maladies du Cœur et des Vaisseaux*, **66**, 1027.
- Wellens, H. J. J. (1975). Contribution of cardiac pacing to our understanding of the Wolff-Parkinson-White syndrome. *British Heart Journal*, **37**, 231.
- Wellens, H. J. J., and Durrer, D. (1973). Combined conduction disturbances in two AV pathways in patients with Wolff-Parkinson-White syndrome. *European Journal of Cardiology*, **1**, 23.
- Wellens, H. J. J., and Durrer, D. (1975). The role of an accessory atrioventricular pathway in reciprocal tachycardia: observations in patients with and without the Wolff-Parkinson-White syndrome. *Circulation*, **52**, 58.
- Wellens, H. J. J., Schuilenburg, R. M., and Durrer, D. (1971). Electrical stimulation of the heart in patients with Wolff-Parkinson-White syndrome, type A. *Circulation*, **43**, 99.
- Wit, A. L., Weiss, M. B., Berkowitz, W. D., Rosen, K. M., Steiner, C., and Damato, A. N. (1970). Pattern of atrioventricular conduction in the human heart. *Circulation Research*, **27**, 345.
- Wu, D., Denes, P., Dhingra, R., and Rosen, K. M. (1974). Nature of the gap phenomenon in man. *Circulation Research*, **34**, 682.
- Zipes, D. P., Rothbaum, D. A., and DeJoseph, R. L. (1974). Pre-excitation syndrome. *Cardiovascular Clinics*, **6**, 209.

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